The Effect of Dietary Sodium on Right Ventricular Failure-induced Ascites, Gain and Fat Deposition in Meat-type Chickens

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ABSTRACT

Experiments were carried out using various levels of sodium (Na^+) from NaCl or $NaHCO_3$ to determine: 1) the level of Na^+ required to induce ascites alone or in combination with cold temperature and 2) the effect of Na^+ on weight gain and fat deposition in broiler chickens. In experiment 1, there were no cases of ascites using levels of Na^+, from NaCl at 0.14% to 0.44% in the feed from day 3 or using added Na^+, from NaCl at 0.0% to 0.12% in the water from day 3 with a level of 0.14% in the feed. There was no significant difference in 21 or 42 day body weight, feed conversion, or right ventricle:total ventricle (RV:TV) ratio between treatment groups. Day 3 to 4 body weight gains were significantly increased in all treatment groups with added Na^+ ($p < 0.01$).

In experiment 2, with levels of added Na^+, from NaCl, at 0.0% to 0.12% in the water with a level of 0.20% in the feed there were two cases of ascites, one at day 7 and one at day 40 at the 0.12% level. There were no significant differences in body weight at days 21 or 42 or in the RV:TV ratios between groups. Feed conversions were improved ($p < 0.01$) with the lowest and highest levels of Na^+ and a significant increase in day 3 to 4 body weight gain, with increasing Na^+ in all treatment groups, was observed.

In experiment 3, levels of added Na^+ with NaCl at 0.135% and with $NaHCO_3$ at 0.145% in the water, with a level of 0.20% in the feed, induced six cases of ascites in the NaCl group and 29 cases of ascites (20.7%) in the $NaHCO_3$ group through day 35. At day 35, pen temperature was decreased to 12°C. Between days 35 and 56 there were five cases of ascites in the control group, 15 in the NaCl group, and 29 in the $NaHCO_3$ group.

There was an increase in mortality from ascites, in day 3 to 4 body weight, and a decrease in day 42 body weight with increased Na^+ in the NaCl and $NaHCO_3$ groups. There were no significant differences in day 56 feed conversion or body weight, carcass weight, chilled carcass weight or abdominal fat-pad weight. It is concluded that:
1. Excess dietary Na^+ induces pulmonary hypertension in broiler chickens, as measured by an increased RV:TV ratio, and Na^+ in water is more toxic than in feed although feed and water Na^+ are additive.
2. There is a "threshold" at approximately 0.12% Na^+ in drinking water, with 0.20% in the feed, above which there is a marked increase in Na^+ toxicity in broiler chickens.
3. Cold and dietary Na^+ are additive in producing pulmonary hypertension, in broiler chickens.

RÉSUMÉ

Des expériences ont été effectuées utilisant divers niveaux de sodium (Na^+) sous forme de NaCl ou de $NaHCO_3$ afin de déterminer : 1. le niveau de Na^+ requis pour provoquer des ascites, seul ou en combinaison avec les températures froides, et 2. l'effet du Na^+ sur le gain de poids et la déposition de graisse chez des poulets de gril. Dans l'expérience 1, il n'y eut aucun cas d'ascite utilisant, à partir du jour 3, des niveaux de Na^+ allant de 0,14 % à 0,44 % de NaCl ajouté aux rations ou bien de 0,0 % à 0,12 % de Na^+ dans l'eau avec 0,14 % dans les rations. Il n'y eut aucune différence significative de poids corporel, de conversion alimentaire ou de rapport ventricule droit à ventricules totales (VD:VT) entre les groupes expérimentaux après 21 ou 42 jours. Les gains de poids corporel étaient significativement plus élevés chez tous les groupes expérimentaux Na^+ ajouté aux jours 3 à 4 ($p < 0,01$).

Dans l'expérience 2, avec des niveaux de Na^+ ajouté allant de 0,0 % à 0,12 % de NaCl dans l'eau et un niveau de 0,12 % dans les rations, il s'est produit deux cas d'ascite, l'un au jour 7 et l'autre au jour 40 au niveau 0,12 %. Il n'y eut aucune différence significative de poids corporel aux jours 21 ou 42 ou dans les rapports VD:VT entre les groupes. La conversion alimentaire était améliorée ($p < 0,01$) avec les niveaux les plus faibles et les plus élevés de Na^+ et des augmentations significatives du gain de poids corporel aux jours 3 à 4 ont été observées avec des niveaux croissants de Na^+ chez tous les groupes expérimentaux.

Dans l'expérience 3, les niveaux de Na^+ ajouté, avec 0,135 % de NaCl et 0,145 % de $NaHCO_3$ dans l'eau et 0,20 % dans les rations, ont provoqué 6 cas d'ascite dans le groupe NaCl et 29 cas d'ascite (10,7 %) dans le groupe $NaHCO_3$ jusqu'au jour 35. Au jour 35, la température a été diminuée à 12 °C. Entre les jours 35 et 56 il s'est produit 5 cas d'ascite chez le groupe témoin, 15 chez le groupe NaCl et 29 chez le groupe $NaHCO_3$.

Chez les groupes NaCl et $NaHCO_3$, il s'est produit une mortalité plus élevée résultant d'ascite, une augmenta-

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tion du poids corporel aux jours 3 à 4 et une diminution du poids corporel au jour 42. Il n'y avait aucune différence significative dans la conversion alimentaire ou le poids corporel, poids de carcasse, poids de carcasse refroidie, ou poids des tissus adipeux abdominaux au jour 56. Nous en concluons que : le Na^+ alimentaire en excès provoque une hypertension pulmonaire chez les poussins de gril, telle que mesurée par un rapport VD:VT accru, et le Na^+ dans l'eau est plus toxique que dans les rations, quoique le Na^+ dans les aliments et l'eau sont additifs.

Il y aurait un seuil toxique qui se situerait à approximativement 0,12 % de Na^+ dans l'eau de boisson, avec 0,20 % dans les rations, au-dessus duquel il y a une augmentation prononcée de la susceptibilité au Na^+ des poussins de gril. Le froid et le Na^+ alimentaire ont des effets additifs sur la production d'hypertension pulmonaire chez les poussins de gril. *(Traduit par Dr Bernard Delorme)*

INTRODUCTION

The evidence that increased dietary sodium (NA^+) can cause ascites in chickens goes back many years (1–7). However, it has been shown recently that levels of Na^+ lower than previously reported result in ascites in broiler chickens (8). Corticosteroids also cause ascites in chickens, probably by inducing Na^+ retention (9,10). Although heart changes have been noted in Na^+-induced ascites, the literature suggests that these were caused by hypertension or were secondary, and that ascites and edema were the result of kidney disease or Na^+ induced oncotic differences allowing excess fluid to leave the small vessels and collect in the tissues and body cavities (1,10–12). Recent research has shown that ascites related to high dietary Na^+ is caused by increased hydraulic pressure in the venous system following right ventricular failure from valvular insufficiency as a result of dilation and hypertrophy of the right ventricle in response to pulmonary hypertension (PH) (8). In chickens, right ventricular hypertrophy is a direct response to PH (13–15). PH may be caused by a variety of factors that increase blood flow or increase resistance to flow in the lung (8). Na^+ may cause PH in meat-type chickens by increasing blood volume or decreasing red blood cell deformability (8). Cold causes PH by increasing blood flow (16) but the combined effect of cold and Na^+ have not been investigated. Moderate levels of dietary Na^+ improve weight-gain in broilers, likely by increasing water consumption (17,18). In addition, Na^+ causes water retention, augmenting weight-gain (8). The objectives of this research were to: confirm the result of a previous study that showed that moderate levels of dietary Na^+ caused PH-induced ascites (8); assess the additive effect of cold with increased Na^+; compare the effect of increased Na^+ in feed and water; compare Na^+ from NaCl and $NaHCO_3$.

MATERIALS AND METHODS

EXPERIMENT 1

Nine hundred and sixty wing-banded, commercial, day-old male broilers (Arbor-Acre strain) were randomly assigned to 24 pens in two rooms. All treatment groups were fed commercial broiler starter to day 21, grower to day 35 and finisher to day 46 which was the same except for differences in Na^+ level. All broilers were on the control diet and tap water (containing 0.0024% Na^+) for 48 h. On day 3, three replicates of each of four treatment groups in room 1 were offered tap water and feed containing 0.14, 0.24, 0.34, or 0.44% Na^+. In room 2, all birds received the same feed (0.14% Na^+) and water containing different levels of added Na^+ from NaCl (0.0, 0.03, 0.06, 0.12%). Each pen contained 40 birds, and brooding and general management were according to industry practice. The broilers were weighed weekly from day 1 and feed conversions were calculated. Birds were also weighed on days 3 and 4 to determine weight increases due to water retention. All broilers that died were necropsied. Broilers were processed on day 47 and hearts collected for right ventricle: total ventricular (RV:TV) ratio. Atria, major vessels and gross fat were cut off and the right ventricle (RV) was cut away from the left ventricle and septum. The RV was weighed and the left ventricle and septum were added to obtain total ventricle (TV) weight. The RV:TV ratio was computed.

EXPERIMENT 2

Seven hundred and eighty wing-banded commercial, day-old male broilers (Arbor-Acre strain) were randomly assigned to 12 pens in room 1. The treatment was the same as room 2, in experiment 1, except that Na^+ in the feed was at 0.20% and five broilers from each pen were examined on day 15, 22, 28 and 36 for evidence of Na^+ induced heart and lung changes.

EXPERIMENT 3

Four hundred and twenty wing-banded day-old commercial male broilers (Arbor-Acre strain) were randomly assigned to 12 pens in room 1. All broilers were fed commercial broiler starter to day 21, grower to day 35 and finisher to day 56. All diets contained 0.20% Na^+. All broilers received tap water containing 0.0025% Na^+ for 48 hours. On day 3, four replicates of each of three treatment groups were offered different levels of added Na^+ in the drinking water [group A, 0.0% (control group); group B, 0.135% Na^+ from NaCl; group C, 0.145% Na^+ from $NaHCO_3$ (Na^+ from NaCl and $NaHCO_3$ was calculated to be 0.135% but analysis of drinking water consistently gave 0.145% of Na^+ with $NaHCO_3$)]. Broilers were weighed weekly from day 1 and feed conversions were calculated. On days 3 and 4 birds were weighed to determine the average weight gain. On day 35, room temperature was decreased to 12°C. Broilers were processed on day 57. Dry carcass, fat pad and wet carcass weights were determined. Hearts were retained for RV:TV evaluation. All broilers that died were necropsied. Histological examinations of heart, lung, kidney, liver and testes were carried out on a small number of broilers that died from ascites before day 21.

Diets were prepared at the Arkell Poultry Research Station and were similar to corn and soyabean based commercial broiler feed. Crude protein (percent) and metabolizable energy (Kcal/kg) were: starter, 22 and 3074; grower, 20 and 3129; finisher, 18 and 3200. Sodium in the feed was from NaCl. The actual sodium levels of feed and water were determined by analysis using atomic emission spectrometry.

STATISTICAL ANALYSIS

In experiment 1 data from each room were analyzed separately with treatments randomized over the 12 pens in each room. Data for the response variables three to four day body weight gain, 21 and 43 day body weight and RV:TV ratio were subjected to an analysis of variance procedure. Those response variables having a significant F-test were further analyzed by Duncan's Multiple Range Test.

Data from experiment 2 consisted of the same response variables which were analyzed in the same manner as experiment 1. Experiment 3 data included the same response variable as experiments 1 and 2 as well as 56 day body weight and carcass characteristics. The weekly percentage of birds dying of ascites for each treatment group was calculated.

The procedures for animal experimentation were approved by the Animal Care Committee of the University of Guelph.

RESULTS

Tables I and II tabulate the body weight and RV:TV data from experiment 1. There were no cases of ascites using levels of Na^+, from NaCl at 0.14% to 0.44% in the feed from day 3 or using added Na^+, from NaCl at 0.0% to 0.12% in the water from day 3 with a level of 0.14% in the feed. There were no significant differences in body weight at days 21 and 42, or feed conversion, or RV:TV ratio between treatment groups although body weight was lower at the 0.14% Na^+ level in both trials. Day 3 to 4 weight gain was significantly increased in all treatment groups with added Na^+ ($p<0.01$).

Table III tabulates the body weight, feed conversion and RV:TV data for experiment 2, using levels of added Na^+ from NaCl at 0.0% to 0.12% in the water with a level of 0.20% in the feed. There were two cases of ascites, one at day 7 and one at day 40 at the 0.12% level. There was no significant difference in body weight at days 21 or 42 or in RV:TV ratio between groups.

TABLE I. Effect of feed sodium on 3 to 4 day weight gain, 21 and 42 day body weight and RV:TV ratio at 43 days (Experiment 1)

	Treatment group					
	1	2	3	4		
Feed Na^+	0.14%	0.24%	0.34%	0.44%	±SD	SIG
Response variable						
1. Body weight gain (g)						
3–4 days	15.2^d	19.0^c	21.2^b	22.8^a	0.76	**
2. Body weight						
21 days	712	738	732	750	13.7	NS
42 days	2143	2184	2159	2198	54.9	NS
3. RV:TV (43 days)	0.216	0.210	0.216	0.217	0.01	NS

$^{a-d}$Means followed by different letters are significantly different ($p<0.01$)
NS = Nonsignificant ($p>0.05$)
SIG = Significance

TABLE II. Effect of water sodium content on 3 to 4 day weight gain, 21 and 42 day body weight and RV:TV ratio at 43 days (Experiment 1)

	Treatment group					
	1	2	3	4		
Feed Na^+	0.14%	0.14%	0.14%	0.14%		
Water Na^+	0.0%	0.03%	0.06%	0.12%	±SD	SIG
Response variable						
1. Body weight gain (g)						
3–4 days	15.5^c	18.0^b	20.5^a	22.3^a	1.15	**
2. Body weight						
21 days	707	729	712	726	28.5	NS
42 days	2186	2246	2225	2212	31.0	NS
3. RV:TV (43 days)	0.218	0.213	0.219	0.214	0.004	NS

$^{a-c}$Means followed by different letters are significantly different ($p<0.01$)
NS = Nonsignificant ($p>0.05$)
SIG = Significance

TABLE III. Effect of water sodium on 3 to 4 day weight gain, 21 and 42 day body weight, feed conversion, and RV:TV ratio (Experiment 2)

	Treatment group					
	1	2	3	4		
Feed Na^+	0.20%	0.20%	0.20%	0.20%		
Water Na^+	0.0%	0.03%	0.06%	0.12%	±SD	SIG
Response variable						
1. Body weight gain (g)						
3–4 days	17.1^d	18.5^c	19.6^b	22.5^a	0.40	**
2. Body weight						
21 days	733	734	744	721	20.5	NS
42 days	2259	2200	2231	2188	40.3	NS
3. Feed intake: body weight gain						
0–42 days	1.88^b	2.01^a	2.00^a	1.91^b	0.04	**
4. RV:TV (43 days)	0.228	0.226	0.226	0.230	0.01	NS

$^{a-d}$Means followed by different letters are significantly different ($p<0.01$)
NS = Nonsignificant ($p>0.05$)
SIG = Significance

Feed conversion was improved ($p< 0.01$) with the lowest and highest levels of Na^+ and there was a significant increase in day 3 to 4 body weight gain with increasing Na^+ in all treatment groups. No lesions were present in the heart or lungs or broilers examined on days 15, 22, 28 or 36.

Tables IV and V tabulate the results from experiment 3. Levels of added Na^+ from NaCl at 0.135% and from $NaHCO_3$ at 0.145% in the water with a level of 0.20% in the feed, induced six cases of ascites in the NaCl group and 29 cases of ascites in the $NaHCO_3$ group to day 35. At day 35 pen temperature was dropped to 12°C. Between days 35 and 56 there were five cases of ascites in the control group, 15 in the NaCl group and 29 in the $NaHCO_3$ treated group.

There was an increase in mortality from ascites (Table IV) and in day 3 to 4 body weight gain, although gain

TABLE IV. Ascites mortality by week in three groups of broiler chickens offered different levels of Na^+ in the water from day 3. Feed contained 0.20% Na^+. Pen temperature was reduced to 12°C at day 35 (Experiment 3)

Day	1–7	8–14	15–21	22–28	29–35	36–42	43–49	50–56	Total ascites	%
Group A 0.0% Na^+	0	0	0	0	0	0	2	3	5	3.6
Group B 0.135% Na^+ (NaCl)	0	3	0	0	3	4	7	4	21	15.0
Group C 0.145% Na^+ ($NaHCO_3$)	2	12	9	4	2	13	10	6	58	41.4
Total ascites	2	15	9	4	5	17	19	13	84	

TABLE V. Effect of sodium as NaCl and $NaHCO_3$ given in drinking water on weight gain, feed conversion, carcass characteristics and RV:TV ratio in broiler chickens (Experiment 3)

	Group A	B	C		
Feed Na^+	0.20	0.20	0.20		
Water Na^+	0.0%	0.135% (NaCl)	0.145% ($NaHCO_3$)	±SD	SIG
Response variable					
1. Body weight gain (g)					
3–4 days	17.1[c]	26.3[a]	20.7[b]	1.4	**
2. Body weight (g)					
21 days	712	715	666	27.8	NS
42 days	2148[a]	2088[ab]	2028[b]	54.8	**
56 days	3082	3013	3016	71.5	NS
3. Feed intake: body weight gain 0–56 days	2.17	2.19	2.15	0.08	NS
4. Carcass wt (g)	2161	2099	2082	77.4	NS
5. Chilled carcass wt (g)	2335	2252	2239	67.3	NS
6. Abdominal fat pad (g)	69.8	62.9	57.9	6.5	NS
7. RV:TV (57 days)	0.242	0.236	0.259	0.01	NS

[a-c]Means followed by different letters are significantly different ($p<0.01$)
NS = Nonsignificant ($p>0.05$)
SIG = Significance

was less with $NaHCO_3$ than with NaCl. There was a decrease in day 42 body weight with 0.145% Na^+ as $NaHCO_3$ in group 3 (Table V). There was no significant difference in feed conversion at day 56 or in day 21 or 56 body weight, carcass weight, chilled carcass weight or abdominal fat-pad weight (Table V).

Broilers which died from PH-induced ascites had marked hypertrophy of the RV wall (mean RV:TV ratio 0.394) compared to broilers that died from other causes (mean RV:TV ratio 0.217). There was no difference in mean RV:TV ratio in broilers which survived. Several of the broilers that died from PH before day 21 had testicular edema because of cystic dilation of seminiferous tubules as described by Sillar *et al* (19). Mortality from Na^+ induced ascites was typical of field cases with the highest incidence in the first 14 to 21 days and a lower but continuing incidence after day 21.

DISCUSSION

Sodium is not usually toxic unless it is present at such high concentration that the kidney receives more Na^+ than water required for excretion (20). However birds have poor renal concentrating ability and have difficulty reducing plasma osmolality by excretion of salt in excess of water (21). The increased susceptibility to Na^+ in the drinking water in broiler chickens prior to day 21 may be related to an insufficiently developed kidney unable to excrete Na^+ with the efficiency found in older chickens. In chickens a rise in plasma osmolality and/or Na^+ concentration stimulates plasma vasotocin (AVT) (22). AVT functions as an antidiuretic hormone and might further reduce Na^+ excretion.

Sodium levels in feed are usually in the 0.14% to 0.30% range (0.35 to 0.75% NaCl), although higher levels are used by some companies because high levels of Na^+ are said to increase growth rate. Na^+ from sources other than NaCl may be present in feed and Na^+ levels are occasionally above 0.30%.

Sodium in the drinking water is more harmful than in feed at the same concentration because broiler chickens consume by weight 1.5 to 2.5 times more water than feed, and Na^+ in feed or water increases water consumption. The effect of sodium in feed and water is additive and Na^+ from all nutrient sources may be additive in chickens (1). Ascites has been frequently reported in poultry from increased Na^+ levels in feed and water (1,2,3,5, 6,10) but ascites did not occur at Na^+ levels of 0.44% (approx. 1.10% NaCl) in the feed in experiment 1.

Pulmonary hypertension is caused by increased blood flow or increased resistance to blood flow in the lung (23). Sodium could increase blood flow by expanding blood volume and the significant day 3 to 4 body weight gain in chickens on increased Na^+ would suggest that there is an increase in blood volume as there is in rats (24). In rats NaCl induces volume expansion while $NaHCO_3$ does not (25). $NaHCO_3$ produced less day 3 to 4 body weight gain than NaCl indicating that increased blood volume was not the reason for the increase in ascites with $NaHCO_3$. High dietary Na^+ increases body temperature probably by altering the hypothalamic thermoregulatory set-point (26). This would increase the body oxygen requirement and increase blood flow through the lung (7). Increased interstitial fluid in the lung from Na^+-induced fluid retention could compress blood capillaries and increase resistance to blood flow. Sodium affects red blood cell rigidity and size and could increase resistance to flow in the small blood capillaries in the lung (8,18,27,28). Increased dietary

Na^+ might also cause arteriolar vasoconstriction as it does in some humans resulting in increased resistance to flow (29,30). In mammals excess sodium does not result in PH and right ventricular failure as it does in poultry although it can be involved in left ventricular hypertrophy in both birds and mammals (8,24,31). Sodium toxicity in mammals causes dehydration and diarrhea unless it is associated with water deprivation, in which case nervous signs may occur. These forms of Na^+ toxicity are reported in chickens at or above 0.45% Na^+ in the water (1,7) or when high levels of Na^+ are present in feed, particularly when water is not freely available (4).

In field outbreaks of ascites in chickens caused by high dietary Na^+, the Na^+ may be from feed due to mixing error, from ingredients containing high levels of Na^+ (2), or the Na^+ may be in the drinking water. Sodium may be deliberately added to water when an ion-exchange water softener is used, or the natural water supply may contain excess Na^+ as it frequently does in western Canada. In the field, water Na^+ levels much lower than those reported here (0.02 to 0.12% in the water) may cause ascites in broiler chickens (Julian RJ, unpublished data). Excess Na^+ may cause wet droppings and a slight increase in mortality from ascites. Both sexes are affected equally (23). At higher levels of Na^+ (above 0.12%) there is a marked increase in the mortality from ascites in young broilers and a low incidence of stunting may occur. High Na^+ (above 0.24%) in the water may cause severe, acute illness with dehydration, nervous signs and death (23).

It is difficult to be sure whether the marked increase in ascites in group C ($NaHCO_3$) of experiment 3 is entirely from the very small increase in Na^+, from 0.135% in group B (total ascites 15%) to 0.145% in group C (total ascites 41%), or whether $NaHCO_3$ has an additional effect. $NaHCO_3$ is less toxic in humans and rats (25) apparently because it causes less blood volume increase. Both NaCl and $NaHCO_3$ have been reported to cause kidney lesions in chickens (1,7,10,11, 32). Kidney lesions were not present at the levels of Na^+ used in this experiment.

Cold is a very important cause of PH-induced ascites both at low and high altitude. Cold increases PH because it increases blood flow by increasing the oxygen requirement (16). It would appear that whatever mechanism is involved in Na^+-induced PH, it adds to the problem of increased blood flow caused by cold temperature since there was a marked increase in ascites mortality in groups B, 15 and C, 29 compared to group A, 5 after the pen temperature was dropped to 12°C at day 35.

Increased dietary Na^+ is reported to increase weight gain and feed conversion (1,8,17,18,32). This may be because it increases water and feed consumption or it may be the effect of water retention. High levels of Na^+ reduce growth (1,7) as occurred at day 42 in the $NaHCO_3$ group. The most Na^+ sensitive chickens died throughout the trial which would influence the day 42 and 56 results. Although the difference was not significant, increasing dietary Na^+ appeared to reduce carcass fat in experiment 3 (Table V).

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